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Corrigendum

Corrigendum to "The dark sides of amyloid in Alzheimer's disease pathogenesis" [FEBS Lett. 588 (5) (2014) 641–652]

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On page 642, paragraph 2, right column, line 12 the following reference should be inserted:

[170]. Sorrentino, G. and Bonavita, V. (2007) Neurodegeneration and Alzheimer's disease: the lesson from tauopathies. Neurol. Sci. 28, 63–71.

The first 19 lines of paragraph 4.2, on page 645, should read:

"APP seems to have a role in neural degeneration mostly as it is processed, rather than through Aβ formation [15]. For instance, there is now broad evidence of the neuroprotective and neuroproliferative role of sAPPα in adult neuron [92,93]. Moreover, mice lacking APP show a smaller brain size [94], and sAPPα expression alone reverses this effect [95]. Furthermore, it has been showed that ERK-induced axon growth is prompted by sAPPα and sAPPβ (generated in a 9:1 ratio) and that amino terminal fragments of APP can shift stem cell growth toward a neural phenotype [96]. Overall, all this evidence seems to suggest that APP processing might be an important inductor of neural stem cell differentiation [15] (Fig. 2)."

Lines 19–42, on page 646, paragraph 6.0, should read:

"The association between AD and ApoE is documented by observations of structural variants of the ApoE protein [133]. In humans, ApoE4 expression increases as a consequence of neuronal damage and a neuron-specific proteolysis, guided by astrocytes, takes place [151,152]. More specifically, ApoE4, cut by ApoE Cleaving Enzyme (AECE) and missing the 272–299 residues (D272–299), is not processed by the secretory pathway as it translocates to the cytosol [133]. On the other hand, D272–299-ApoE4 is integrated in the neurofibrillary tangle-like structures [152]. Accordingly, mice overexpressing cleaved-ApoE4 die within 4 months as they form AD-like neurofibrillary tangles. Furthermore, when mutated ApoE is expressed at lower levels, learning and memory deficits are observed at 6–7 months [153]. In addition, in Neuro-2a cell line, the D272–299-ApoE4 impairs complex III and IV respiratory functions [154], and mice expressing the same variant of ApoE display impaired axonal transport, with mitochondria accumulation in bulb-like dilations [133,155]."

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